CASE REPORTS

Constricting Exercises to Correct Postoperative Fecal Incontinence

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SOME degree of fecal incontinence is a distressing occasional aftermath of operations that involve the cutting of the sphincter ani, and operations performed with the intent of curing it may be unsuccessful.

The following report illustrates a method of treatment which enabled a patient so afflicted to regain complete control.

The patient, a young woman, had been operated upon for perirectal infection. Fecal incontinence had ensued, and another operation had not relieved it. Consequently, restraining opiates had been given and the patient was taking them when examined by the author.

Three fingers could be readily introduced into the anal canal. Upon them the patient could exert a feeble circular pressure. She was told that some of the circular fibres of the sphincter were still intact and that, just as a prizefighter could train and strengthen his hitting muscles by hitting exercises, so she could train and strengthen these fibres by constricting exercises.

The largest of a set of Young's anal canal dilators was introduced. This the patient could just retain. She was given the dilator and instructed she should, while lying down, introduce it several times a day, and each time try intermittently for a few minutes to grip it. She was informed she would soon be able to do this, and that then she would be given a smaller dilator and so on gradually until she could grip one of the smallest diameter. The patient followed the procedure conscientiously and in a few weeks reported that she had regained control except when walking.

Directed to stand up, to put her heels together, to separate widely the toe regions, and then to contract her buttock muscles, she at once felt the added protection. She was advised to walk with the feet thus splayed and while walking to practice the constricting exercises. In a few weeks the incontinence was completely corrected.

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Erythermalgia

Report of a Case, and Response to a New Therapeutic Approach

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SINCE the original report of erythermalgia by Weir-Mitchell in 1872[16] and his subsequent suggestion in 1878[17] that it be named "erythromelalgia," little was done in attempting to discover a rational method of therapeusis till the latter 1930s. In 1937 Mufson[18] suggested that the pronounced vasodilatation seen in this disease might be due to a lack of vasoconstrictor stimuli, and advised the use of epinephrine hydrochloride, either by inhalation or hypodermic administration. This was soon followed by Smith's and Allen's report in 1938[19] in which they recommended that the term "erythermalgia" be substituted for the original name suggested by Weir-Mitchell, and advocated the use of acetylsalicylic acid. They reported pronounced relief from the use of 0.65 gm. four or five times daily, the relief from a single dose sometimes lasting several days. Although no adequate explanation existed for this, as acetylsalicylic acid, except for its analgesic effects, pharmacologically at least, tended toward a greater cutaneous vasodilatation, Allen, Barker and Hines[2] came to regard this phenomenon as almost diagnostic of this disease process. In the light of more recent knowledge, the explanation of this may lie in the fact that salicylates in toxic doses are strong inhibitors of the enzyme hyaluronidase.[20] Although hyaluronic acid is not present in the capillary wall itself,[6, 15] it is known to make up the intercellular cement substance of the supporting connective tissue of these small vessels, and it is because of the weakening of this connective tissue cement substance that capillary damage occurs.[6] Possibly patients with erythermalgia may be more susceptible to this action of acetylsalicylic acid so that only small doses may be necessary to abolish the action of hyaluronidase and thus prevent the extravascular edema with the consequent distressing soft-tissue swelling. Despite the foregoing, however, Wright[22] has stated that in his experience acetylsalicylic acid gives patients only temporary relief and that the results of the administration of epinephrine by either route are far from striking.

Following the lead of Mufson,[18] whose concept of the physiopathology in this disease process seemed to be capable of explaining all the symptoms, it was decided to use a probable adrenalin precursor, which could be taken orally, for the purpose of producing a humoral antihistaminic vasoconstriction. For this purpose the amino acid l(—)-tyrosine together with its co-enzyme precursor, pyridoxine hydrochloride, was chosen as the form of therapy for a patient afflicted with erythermalgia. As there is evidence that tyramine, the amine derived from the amino acid tyrosine, may be identical with sympathin,[7] the rationale for its use seemed even more justified.

CASE REPORT

A white married woman, 30 years of age, was referred for study and treatment on account of a vasodilating type of peripheral vascular disease process, which had been diagnosed as erythermalgia and which she had been aware of since July 1947. The patient said that at that time, for no known reason, she first experienced a hot, and at times burning, throbbing pain in the fingers and toes, which was associated with swelling and reddening of the affected areas. This always followed either exposure of the extremities to heat (hot dishwater) or increased activity of the affected digits (knitting, etc.) and was relieved by immersing the parts in cold water. The swelling and redness would persist for hours after the disappearance of pain, so that the fingers of both hands generally appeared turgid. Because of this she had begun to shun social gatherings, as she was ashamed of the appearance of her hands. The upper extremities had always been much more affected than the lower, and the right upper extremity more than the left. On further questioning she admitted noticing an increasing intolerance to